LETTERS TO THE EDITOR

Regarding "Early carotid endarterectomy in symptomatic patients is associated with poorer perioperative outcomes"

We read with interest this large retrospective study by Rockman et al[1] concluding that carotid endarterectomy (CEA) should be delayed in patients with symptomatic carotid disease. These results disagree with pooled analysis of the European Carotid Surgery Trial (ECST) and North American Symptomatic Carotid Endarterectomy Trial (NASCET), which suggest the benefit is greatest in patients operated on ≤2 weeks of the ischemic event.[2] Data from our center suggest the risk of recurrent ischemia to be 30% in the 7 weeks after initial event (unpublished data). This is an incidence similar to that found by the Oxford Vascular Study Group,[3] where the risk of stroke was 21% at 2 weeks and 32% at 12 weeks, and half of the strokes were disabling or fatal in patients with significant carotid disease.

Furthermore, most of the postoperative strokes in this study were hemorrhagic, which may reflect the natural history of some strokes where delayed intracerebral hemorrhage follows ischemic stroke. Thus, the high postoperative stroke rate reported here may also include anticipated events and the true postoperative stroke rate may in fact be lower.

If we were, therefore, to follow the recommendations suggested by the authors, more patients will have potentially preventable strokes whilst waiting for surgery. The higher risk of perioperative stroke rate in the early CEA group will need to be balanced with the high risk of stroke if surgery is to be delayed.

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Reply

We appreciate the interest and concerns of Toby Richards and colleagues regarding our recent publication, "Early carotid endarterectomy in symptomatic patients is associated with poorer perioperative outcomes."

Our institutional retrospective study found that patients who underwent carotid endarterectomy (CEA) ≤4 weeks of ipsilateral stroke appeared to experience a significantly increased rate of perioperative stroke compared with patients who underwent CEA in a more delayed fashion. Clearly Richards and colleagues are correct that our results were markedly different from a number of randomized, prospective trials that examined this topic, including data from NASCET, the ECST, and the Rothwell study, which were all referenced in our manuscript. These studies, and others, have suggested that early operation after stroke is in fact beneficial when compared with delayed treatment. Other limitations of our data, also acknowledged in our manuscript, included the fact that we did not have information regarding possible recurrent strokes that may have occurred during an arbitrary waiting period before surgery.

The difference in findings between our institutional data and the recent data regarding this topic is one of the reasons we felt compelled to report this real-world experience. With knowledge of the issue and the current literature, the surgeons at our institution are making their best judgments about the proper timing of intervention in individual patients, and the results have nonetheless shown that the stroke patients operated on early had worse outcomes.

Because of the limitations of our study, the authors agree that delayed surgery cannot be uniformly recommended for all stroke patients. However, currently it is not exactly clearly defined which stroke patients will benefit from early surgery and which would be better served with a defined waiting period before revascularization. Hence, our final recommendation was to consider a waiting period of 4 weeks in some stroke patients who are candidates for CEA, based on the surgeon's judgment in each individual patient and situation.

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Regarding "Massive spouting bleeding from chronic stasis ulceration caused by arteriovenous communication of the lower extremity"

I would like to congratulate the authors Komai et al (J Vasc Surg 2006;44:658-9) for their successful management of potentially life-threatening—if not limb-threatening—venous bleeding. I wholeheartedly agree with their opinion as well as their recommendation. However, I would like to remind the authors and readers together that such massive/recurrent bleeding from the varicose vein that they experienced should arouse the suspicion of a congenital vascular malformation (CVM) as its hidden cause.[1] Although its etiology could be arguable, an arteriovenous malformation (AVM) should be considered as the cause of an AV communication until proven otherwise.

The arteriogram of this case (Fig 3) has shown early venous phase, suggesting substantial shunting of arterial blood to the venous system. But because of the quality/clarity of the photo (3, *A*), it is very difficult to confirm whether the lesion has a direct communication between artery and vein, defined as a "truncular" lesion, or through the nidus, defined as an "extratruncular" lesion of AVM.[2] The differentiation of these two different types of AVM is extremely important in terms of a prognostic and recurrence point of view.[3]

If it were the truncular lesion in the latter stage of embryogenesis that had developed, a simple embolization of its feeding artery, as was done in this case, should be safe and may be the right answer for the lesion, with the chance of a cure. But if it were the extratruncular lesion in the earlier stage of development, that